Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

Meeting June 26th, 1889.

A DESCRIPTION OF TWO CHINESE BRAINS, AND A NOTE ON THE PLI DE PASSAGE INFERIEURE IN THE HUMAN BRAIN.

Dr. F. X. DERCUM, of Philadelphia, presented two contributions with these titles. For original papers, see page 421.

He exhibited the brains described. Dr. Mills had made a morphological analysis of one in 1886, and descriptions of three Chinese brains had been added to literature by Moritz Benedikt. The six brains thus far analyzed exhibited unusual complexity due to excessive sinuosity of the gyri, and a tendency to excessive transverse fissuration. The frontal lobes were especially large and complex. There was unusual confluence of fissures, indicative of a low degree of development, such as was often seen in the negro's and sometimes in the white man's brain. There was unusual length of the parallel and Sylvian fissures, also eversion of the orbital and temporal lobes.

Dr. MILLS said it was very important to keep adding to our morphological descriptions of such brains until accurate deductions could be drawn from a very large number.

Dr. SPITZKA stated that the dictum that a tendency to confluence of fissures indicated a low type of development was not accepted by most anthropologists. The more brachycephalic the skull, the greater was the development of transverse secondary folds. The elephant, which stood among the highest of mammals, had a broad skull and a tendency to confluence of fissure. The Chinese were not low in development. The internal *pli de passage* in the negro and idiot was significant only when the general cortical development was poor. The real cause of the location of the chief sulci lay in the inherent architecture of the brain, and was due to the arrangement of groups of cells and fibres, but the secondary and tertiary sulci might be influenced by other factors,such as the course of vessels. While we might never be able to localize the higher functions of the mind, there was something in the physiognomy of the Chinese brain which struck him as differing altogether from that of the Caucasian. But it was not the same difference as was noticeable between the negro and Caucasian brains. The peculiar moral attitude of the Chinese pointed to some relation between the structure of their brains and the character of their minds. Still, he had observed a great difference between these two Chinese brains, and there was no feature in them which might not be reproduced in the Caucasian brain.

Dr. DERCUM had not wished to imply that transverse fissuration was indicative of a low type, but thought the unusual confluence showed a lack of cortical development. He did not consider them brains of a low type, but many features such as were found in the brains of the negro and ape were found here also. The excessive sinuosity, however, indicated a higher cortical development. For instance, in one brain there were five well-developed frontal gyri.

Dr. SPITZKA had the same idea as Dr. Dercum. As compared with the Caucasian brain, the Chinese was in some respects inferior, and in others exhibited a superabundance of gyri. He spoke of the resemblance between the negro and ape brains. The difference between the brains of the orang-outang and chimpanzee were greater than those between the brains of the chimpanzee and a human being. There was no uniform appearance of the ape's brain. Vogt and another had classified the human brain into three types—the negro, Mongolian, and Caucasian—corresponding somewhat to the three lower types of the gorilla, chimpanzee, and orang-outang.

Dr. M. Allen Starr, of New York, then read a paper entitled

THE PATHOLOGY OF SENSORY APHASIA, WITH AN ANALYSIS OF FIFTY CASES IN WHICH BROCA'S CENTRE WAS NOT DISEASED.[1]

In questions regarding the localization of cerebral functions, the final appeal must always be made to pathology. And since it is the practical application of the principle to the removal of disease from the human brain which is the chief object in view, its paramount importance cannot be denied. The collection and analysis of reliable and well-observed cases of disease in man is therefore of value, and is the necessary preliminary to surgical therapeutic procedure.

In the study of aphasia and in the localization of the various functions which take part in the use of language, clinical observation has always outrun pathological data. What is the present status of the pathology of aphasia?

It will be admitted that no doubt exists at present in regard to the pathology of motor aphasia. Facts in accord with the dictum of Broca, that a lesion of the posterior part of the third frontal convolution on the left side in right-handed, and on the right side in left-handed, persons produces a loss of the power of using language without any disturbance in the power of understanding words, are too well known and too numerous to require more than a simple statement. And the additional fact that the same effect, though usually but temporary, may follow destruction of the motor speech tract from Broca's centre to the motor nuclei of the pons and medulla rests upon positive data.[2] In the latter case correlated symptoms may enable the position of the lesion to be recognized during life, these symptoms being such as are usually produced by foci of disease in the internal capsule, crus cerebri, or pons varolii.

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[1] Published in full in *Brain*, part xlv.

[2] Raymond et Artaud, Arch. de Neurol., 1883, No. 20.

In regard to the pathology of sensory aphasia, the number of positive facts is by no means large. A careful search through medical literature of the past twenty years has resulted in a collection of fifty cases of aphasia of a distinctly sensory variety, which may be utilized for conclusions, and which have been tabulated. These cases will bear rigid inspection, and conform to Nothnagel's requirements in being cases of limited softening, of long duration, and thoroughly examined both clinically and pathologically.

Conclusions :

(1.) In all of these cases some form of sensory aphasia was present, and in all the lesion lay in the posterior lower third of the brain. The convolutions were found affected in the following order :

First temporal,	in	38	cases.
Second "	"	27	"
Inferior parietal,	"	21	"
Angular gyrus,	"	25	"
Supra marginal gyrus,	"	12	"
Occipital lobe,	"	19	"

In seven of the cases pure word deafness was present. The patients had lost the power to understand speech when heard, though able to read, to talk and to write (Cases III., XVIII., XX., XXX., XXXIII., XXXIV., XL.).

In all of these cases the lesion was limited to the first and second temporal convolutions in their posterior two-thirds.

In eleven of the cases pure word blindness was present. The patients had lost the power to understand words when seen, though able to understand speech and to talk (Cases II., XXIII., XXXV., XYXVI., XXXVII., XXXIX., XLII., XLIII., XLIV., XLVI., XLVII.).

In two of these cases the patients were able to write or copy, but in the remainder they had lost the power or were not tested.

In these cases the lesion was not found uniformly in one location. It affected the angular gyrus in five cases, the occipital lobe in five cases, the temporal convolutions in three cases, the inferior parietal region in three cases, and the supra marginal gyrus in two cases.

By inferior parietal convolutions it is intended to indicate those gyri which lie between the supra marginal gyrus and the angular gyrus, and which are between the interparietal sulcus and the first temporal sulcus, the area lying between P 2 and P 2′ in Ecker's diagram, reproduced in "Ferrier's Functions of the Brain," p. 472.

In twenty-five of the cases the power to recall words and to name objects was impaired. This occurred in some of the cases of pure word deafness and also in some of pure word blindness. In some of these cases the power to recognize the word or name of the object when suggested by another person was preserved. And the lesion in these various cases varied widely, involving any or all of the various gyri included in the sensory speech area, or the subcortical tracts beneath them.

In seven of the cases word deafness and word blindness were present together, and yet the use of language was not lost. The patients could talk (Cases I., VII., XV., XIX., XXIV., XXIX., XXXIV.).

In these cases the lesion lay in the temporal convolutions alone in two cases, and in the remainder it extended posteriorly, involving the inferior parietal, angular and occipital convolutions.

In twenty-seven of the cases word deafness and word blindness were accompanied by more or less impairment in the power to talk. The difficulty in talking in but two cases was a difficulty in the power of pronunciation, such as occurs from lesion of Broca's centre. In all others it consisted of a use of wrong words, or unintelligent phrases, a series of words whose connection was deficient. Paraphasia is therefore the usual accompaniment of sensory aphasia. In these cases the lesion was wide in extent, involving the temporal, parietal and occipital convolutions.

It was impossible to ascertain any constant pathological difference between the cases of sensory aphasia without and with paraphasia. Nor did the power to repeat words after another seem to depend upon the relative situation of the lesion, as might be supposed from Wernicke's assertion that this defect appears with paraphasia when the temporo-frontal tract is involved. For paraphasia with inability to repeat words was found in a few cases where the lesion lay too far back to affect this tract. Paraphasia therefore may be caused by lesions in very various locations.

The analysis of the pathological lesions, therefore, does not bring out as clear a differentiation of the different forms of aphasia as might be desired.

It is evident that word deafness is due to a lesion of the first and second temporal convolutions. It is evident that word blindness may be produced by lesions lying in the region of the inferior parietal lobule, or extending either anteriorly from it into the temporal region or posteriorly into the angular gyrus and occipital lobe. It is evident that these conditions are usually associated, and when occurring together are usually accompanied by paraphasia; in these cases the lesion may lie anywhere within the limits of the sensory aphasic area, which includes the inferior parietal convolutions, the two temporal convolutions and the occipital convolutions.

In the cases here brought together the power of recalling the names of objects was impaired or lost in a large number, and the lesions producing this effect are by no means uniform in position; some lying forward in the tract within the temporal region, others about its middle in the parietal region and in the angular gyrus, others far back in the occipital lobe exclusively. The loss of power to recall the name of objects seen does not therefore indicate very exactly the seat of the disease.

But there are certain additional tests which aid in a more exact localization of verbal amnesia. If the name cannot be recalled because the memory of it is lost, that is, because the physical basis of that memory has been not merely isolated from one connection, but actually destroyed, then it is evident that no other association will suffice to reach it, and that even recognition is impossible.[3] This is the condition in absolute word deafness, which, as we have seen, is due to a lesion in the posterior two-thirds of the first and second temporal convolutions. To fail to recall a word by any means, and to fail to recognize its meaning when heard, is therefore characteristic of a lesion of this area when that lesion is extensive.

But there are cases on record where the power to recall words is impaired while the power to recognize them remains. Cases II., VIII., XXII., XXXVII., XLII., XLIII., XLIV., XLVII. illustrate this condition. This must imply that the auditory memories remain and can be reached through the auditory tract, while the association fibres alone are affected by disease. Now, the lesion in all of those cases lay in the posterior portion of the sensory aphasic area, the temporal convolutions being invaded in only two cases, and in those only at the extreme posterior portion; while in all the inferior parietal convolutions and angular gyrus or occipital lobe were affected. And in all of these cases the lesion was a deep one, invading the white tracts beneath the convolutions; in five of them the lesion was wholly subcortical (Cases II., XXII., XXXVII., XLII., XLVII.).

The conclusion, therefore, appears to be warranted that while failure to recognize a word heard implies destruction of the temporal cortical area, failure to recall the name of an object seen implies destruction of the temporo-occipital association tract in the subcortical white matter.

The latter with the former implies extensive cortical and subcortical disease; the latter without the former implies subcortical disease only, without reference to its extent.[4]

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[3] Attention was first called to this fact by De Watteville, Progrès Médical, March, 1885.

There is a third condition which cannot be passed by in this connection, viz., psychical blindness. If an object is seen but not recognized, it implies that the visual mental image of the object is either destroyed or wholly cut off from its associations. This occurs not infrequently in lesions in the occipital lobe, either unilateral or bilateral. It is very frequently accompanied by the characteristic symptom of a lesion of the occipital lobe, namely, bilateral homonymous hemianopsia.

In twelve of the cases here collected psychical blindness was present. In six of these hemianopsia was also present. In all these cases the occipital lobe was diseased ; twice with the adjacent angular gyrus. Psychical blindness is produced not only by disease in the cortex of the occipital convolutions, but also by disease in the white tracts within the lobe. It is evident, therefore, that the memory pictures of objects lie in the occipital lobe, and a serious loss of them implies a lesion in that region. If the lesion be extensive enough to involve the cuneus, or deep enough to reach the visual tract to the cuneus as it passes beneath the angular gyrus and convexity of the occipital lobe, it will produce hemianopsia. If not, actual blindness may not accompany psychichal blindness. In either case it is found that when things are not recognized, they cannot be named when seen. Hence the symptom of psychical blindness may aid in locating a lesion in the visual-auditory tract, and indicates that the lesion of that tract is in its posterior portion.

We thus have three sets of symptoms which enable us to locate a lesion in the association tract between the occipital and temporal areas, viz., loss of power to recognize the name of an object when heard (word deafness) ; loss of power to recall the name of an object recognized (verbal amnesia) ; loss of power to recognize an object seen whose name is understood (psychical blindness). The first implies a lesion in the temporal end of the tract. The last implies a lesion in the occipital end of the tract. The second implies a lesion between the others, probably beneath the inferior parietal lobule. And reference to the cases cited shows that the clinical distinction is supported by the pathological finding ; that the psychological hypothesis has a confirmation from the facts of disease.

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[4] An interesting clinical example of a lesion in the association tract alone is given by Hughes Bennett in the British Medical Journal, 1828, i. p. 339, case third.

The principle applied to the study of lesions in the visual-auditory tract may be extended to the consideration of other tracts. If word blindness be held, as it justly may, to be merely a variety of psychical blindness, it becomes evident that a distinction must be made between recognizing printed words, recalling printed words, reading aloud, or writing. Hence varieties in the condition of word blindness are possible.

To recognize the meaning of a sign implies integrity in the perceptive process and integrity in the associative process which joins the memory of that sign to some other mental image, giving it meaning. It is the association of two mental images which lies at the basis of any process of understanding. When we see the word "bell," it has a meaning only because the image of the word is associated with that of the object, and a destruction in the process of association will impair at once the power of recognition. It is not surprising, when this is understood, to find that the lesions producing word blindness are situated in various regions. When limited in extent and strictly cortical, the lesion producing word blindness was found in five cases in the angular gyrus and in the cortex immediately anterior to this in the inferior parietal lobule (Cases XXIII., XXXV., XXXIX., XLI., XLVI.). It is here, therefore, that the visual memory pictures are thought to lie. And, in fact, in all the cases in this collection in which the lesion involved this area and in which reading was tested, there was word blindness (twenty-one cases).

The associations between the memory of words seen and the mental image are very numerous, and reach out in different directions. The words *thorn*, *trumpet*, *Madonna*, call up painful, auditory, and visual memories respectively, and one can readily imagine that each of their associations might be impaired without the others being affected. But in all three cases this impairment of association might manifest itself as word blindness. Granting the hypothesis, then, that a lesion of the association tracts will produce word blindness,[5] it is evident that tracts going out in all directions from the angular gyrus as a centre might be invaded, with the result of producing the same symptom. The two chief tracts, those usually tested, will be those to the visual area and to the auditory area. To recall the appearance of an object on seeing its name, and to prononnce the name on seeing the word, are the tests applied to these tracts respectively.

Reading understandingly and reading aloud are, therefore, different processes, the former testing a tract from the angular gyrus backward to the occipital region; the latter testing a tract from the angular gyrus forward, to the temporal region. A loss of the power to read may be associated with lesions in the occipital lobe, as in Cases XXXVII., XLII., XLIII., XLIV., in which the lesion was confined to this region. It may also accompany lesions in the posterior temporal region, which encroach upon the inferior parietal lobule, as in Cases II., X., XII., XXI., XXXVI.—in all of which, though the angular gyrus was intact, the symptom was produced.

The combination of word blindness with word deafness, in temporo-parietal lesions, and the combination of word blindness with psychical blindness and hemianopsia in parieto-occipital lesions appears to be established. Berlin's[6] condition of dyslexia, in which a patient is fatigued unduly by reading, and which he ascribes to a subcortical lesion beneath the angular gyrus, might well be explained by an interference with the association tracts.

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[5] This hypothesis was first broached by De Watteville, Prog. Médical, March, 1885. Freund appears to have overlooked this fact in a recent article, Arch. f. Psych., xx., Ueber optische Aphasie und Seelenblindheit.

[6] Berlin, Eine besondere Art von Wortblindheit, Wiesbaden, 1887.

Another tract which may be tested in this condition is the tract from the angular gyrus to Broca's centre. It is tested by asking a patient to read aloud, a process which may be gone through even though the patient does not understand what is read, as in Cases VII. and XXIII.[7]

Whether this tract is a direct one, or is indirect, via the temporal lobe, is still undecided; and sufficient material is waiting for definite conclusions, though in one of the cases here cited, reading aloud was possible where the temporal lobe was so much injured that word deafness was present. This case would indicate that the tract is a direct one, and if so, it must pass beneath the Island of Reil from behind forward. That it starts from the angular gyrus and passes forward into the inferior parietal lobule and supramarginal convolution is evident from the fact that in all the cases here recorded in which reading aloud was impossible, these parts were invaded by disease.

The last tracts to be tested in connection with printed language are those concerned in writing. Writing spontaneously and copying appear to test the same tract.

In all cases in which these powers were both tested, they were both lost or impaired equally. In all these cases the lesion was in or very near the angular gyrus. This tract starts then from this centre. Its direction and termination are not, however, known, as there are no autopsies upon cases of pure agraphia, excepting in the case of Sigaud (Case XLI.), where the lesion was confined to the angular gyrus and the condition was one of sensory rather than of motor agraphia.

It is evident from this review of the clinical and pathological facts in sensory aphasia that the pathological data warrant a recognition of many of the numerous forms of aphasia recently described. There are aphasias of association as well as cortical aphasias. It is necessary to recognize aphasia from lesion of the visual-auditory or occipito-temporal tract (verbal or auditory amnesia); aphasia from lesions within the occipital lobe giving rise to word blindness with visual amnesia; aphasia from lesions in the tempero-parietal region giving rise to word blindness with word deafness; as well as the simpler forms of cortical aphasia known as word deafness, word blindness, agraphia, and motor aphasia. Thus far the clinical facts rest on pathological findings. Subjective investigation of speech processes, as well as clinical observation, may warrant further distinctions not yet resting on post-mortem records. It is evident that the various possible mental processes involved in memory must be carefully tested in every case; and that small subcortical lesions should not be overlooked.

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[7] A similar case is reported by Hughes Bennett, l. c.

Inasmuch as this investigation of the pathology of sensory aphasia shows the need of more careful examination of aphasics, it may be well to suggest the lines along which such an examination should be made.

It is necessary to investigate:

1. The power to recognize objects seen, heard, felt, smelt or tasted.
2. The power to recall the names of such objects.
3. The power to recognize the names of such objects when heard.
4. The power to call to mind the objects when named.
5. The power to understand speech.

This examination will test the various sensory areas, and especially the temporal convolutions and the association tracts between these convolutions and the different sensory areas. It is also necessary to investigate:

6. The power to understand printed or written words.
7. The power to read aloud and to understand what is read.
8. The power to recall objects whose names are seen.
9. The power to write spontaneously, and to write the names of objects seen, heard, etc.
10. The power to copy and write at dictation.
11. The power to read understandingly what has been written.

These tests will determine the condition of the visual word memories in the angular gyrus, and of the connections between this area and surrounding sensory and motor areas.

It is also necessary to find out whether:

12. The power to speak voluntarily is preserved, and if not, the character of its defects.

13. The power of repeating words after another should also be tested.

DISCUSSION ON DR. STARR'S PAPER.

Dr. LLOYD had now a case of simple homonymous hemianopsia without psychical or word-blindness, and wished to know whether there was any diagnostic importance in this condition as to localization. Was there a preponderance of one hemisphere over another as regarded sensory memories of language?

Dr. STARR said that the matter of psychical blindness had recently been discussed by Wernicke and another. A lesion affecting bilaterally both occipital lobes or the association tract produces psychical blindness, but a lesion along either tract itself does not cause this condition. Nine cases of psychical blindness from lesion of one hemisphere alone had been reported.

AMERICAN ACADEMY OF MEDICINE.

The American Academy of Medicine is endeavoring to make as complete a list as possible of the Alumni of Literary Colleges, in the United States and Canada, who have received the degree of M.D. All recipients of both degrees, literary and medical, are requested to forward their names, at once, to Dr. R. J. Dunglison, Secretary, 814 N. 16th Street, Philadelphia.

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SCALE OR POWDER

☞ SEE "MERCK'S INDEX," PAGES 106 AND 167 ☜